
KIF20A/MKLP2 regulates the division modes of neural progenitor cells during cortical development.

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Public Summary:

Balanced symmetric and asymmetric divisions of neural progenitor cells (NPCs) are crucial for brain development, but the underlying mechanisms are not fully understood. Here we report that mitotic kinesin KIF20A/MKLP2 interacts with RGS3 and plays a crucial role in controlling the division modes of NPCs during cortical neurogenesis. Knockdown of KIF20A in NPCs causes dislocation of RGS3 from the intercellular bridge (ICB), impairs the function of Ephrin-B-RGS cell fate signaling complex, and leads to a transition from proliferative to differentiative divisions. Germline and inducible knockout of KIF20A causes a loss of progenitor cells and neurons and results in thinner cortex and ventriculomegaly. Interestingly, loss of function of KIF20A induces early cell cycle exit and precocious neuronal differentiation without causing substantial cytokinesis defect or apoptosis. Our results identify a RGS-KIF20A axis in the regulation of cell division and suggest a potential link of the ICB to regulation of cell fate determination.

Scientific Abstract:

Balanced symmetric and asymmetric divisions of neural progenitor cells (NPCs) are crucial for brain development, but the underlying mechanisms are not fully understood. Here we report that mitotic kinesin KIF20A/MKLP2 interacts with RGS3 and plays a crucial role in controlling the division modes of NPCs during cortical neurogenesis. Knockdown of KIF20A in NPCs causes dislocation of RGS3 from the intercellular bridge (ICB), impairs the function of Ephrin-B-RGS cell fate signaling complex, and leads to a transition from proliferative to differentiative divisions. Germline and inducible knockout of KIF20A causes a loss of progenitor cells and neurons and results in thinner cortex and ventriculomegaly. Interestingly, loss of function of KIF20A induces early cell cycle exit and precocious neuronal differentiation without causing substantial cytokinesis defect or apoptosis. Our results identify a RGS-KIF20A axis in the regulation of cell division and suggest a potential link of the ICB to regulation of cell fate determination.

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